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CEREBROSPINAL MENINGITIS ("FORAGE POISONING").

By JOHN R. MOHLER, V. M. D., *Chief of the Pathological Division.*

INTRODUCTION.

About 100 years ago (1813) there appeared in Wurttemberg a fatal disease of horses which was termed "head disease" owing to the pronounced manifestation of brain symptoms. The affection spread through certain sections of Europe from 1824 to 1828 and was described as "fever of the nerves." In 1878 the attention of the veterinarians of Saxony was attracted to the disease, which was then termed "nervous sickness," and within the next 10 years it assumed an epizootic character. In fact the malady became so prevalent in and around Borna (near Leipsic, Germany) during the nineties that it became known as the Borna disease. The affection has spread like a plague on two occasions in Belgium, and has also exacted a heavy toll in Russia, Great Britain, Austria-Hungary, and elsewhere. Its appearance in America is by no means of recent occurrence, for the malady was reported by Large in 1847, by Michener in 1850, and by Liautard in 1869 as appearing in both sporadic and enzootic form in several of the Eastern States. Since then the disease has occurred periodically in many States in all sections of the country, and has been the subject of numerous investigations and publications by a number of the leading men of the veterinary profession. It is prevalent with more or less severity every year in certain parts of the United States, and during the year 1912 the Bureau of Animal Industry received urgent requests for help from Colorado, Georgia, Iowa, Kansas, Kentucky, Louisiana, Maryland, Missouri, Nebraska, New Jersey, North Carolina, Oregon, South Carolina, South Dakota, Virginia, and West Virginia. While in 1912 the brunt of the disease seemed to fall on Kansas and Nebraska, other States were also seriously afflicted. In previous years, for instance in 1882 as well as in 1897, the horses of southeastern Texas were reported to have died by the thousand, and

NOTE.—This publication gives information about a serious disease of horses; it is especially suited to veterinarians in the States west of the Mississippi River and in the South.

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in the following year the horses of Iowa were said to have "died like rats." However, Kansas seems to have had more than her share of this trouble, as a severe outbreak that extended over almost the entire State occurred in 1891, while in 1902 and again in 1906 the disease recurred with equal severity in various portions of the State.

NOMENCLATURE.

There has always been considerable discussion and criticism regarding the different names which have been given this malady, and various terms have been applied according as each author in past outbreaks has considered certain symptoms or lesions as the paramount feature of the affection. Thus the disease has been termed "cramp of the neck," "head disease," "mad staggers," "sleepy staggers," etc. Through the recent investigations of Grimm, Schmidt, and others it has been quite definitely established that "head disease," Borna disease, and cerebrospinal meningitis are one and the same, and Hutyra and Marek have accepted this opinion and incorporated it in their "Special Pathology." While at first the Borna disease was considered as a form of cerebrospinal meningitis, the work of Johne and Ostertag (1900) indicated that it was an independent disease, because they failed to find any inflammatory changes in the central nervous system. Accepting this view, Friedberger and Fröhner have separated the two diseases in their "Theory and Practice," basing their differential diagnosis chiefly on the absence of inflammation in the brain and cord of Borna disease. However, since the publication of this excellent work in 1904, Oppenheim, Dexler, Schmidt, and others have shown conclusively that inflammatory lesions are present in the central nervous system, although Dexler has pointed out that in some cases it is necessary to make a systematic examination of a number of slides to discover the inflammatory changes. As a result the more recent writers have adopted the viewpoint that the two terms, Borna disease and cerebrospinal meningitis, are synonymous.

When this disease appeared with such severity in certain sections of the United States in the summer of 1912, there were a number of persons who claimed that it was the Borna disease appearing in the New World for the first time; others diagnosed it as a new horse disease, as influenza, parasitism (due to the palisade worm), paralysis similar to poliomyelitis (infantile paralysis) of man, epidemic cerebrospinal meningitis of man, and equine malaria from the fact that mosquitoes were prevalent and the horses were in lowlands. These erroneous diagnoses, while participated in to a certain extent by some veterinarians, were usually the opinions of physicians, chemists, bacteriologists who were not veterinarians, and others of limited veterinary experience. However, the vast majority of veterinary

practitioners recognized the disease as their old torment—cerebrospinal meningitis, staggers, or forage poisoning.

The latter name came into the literature of the disease as a synonym in 1900 following the investigation of an outbreak by Pearson. He was able to reproduce the disease in experiment horses by feeding them on damaged silage, and by giving them water to drink which had percolated through this silage. Doubtless influenced by the frequent absence of microscopic lesions of the central nervous system, and by the analogy between this disease and meat poisoning of man, Pearson proposed the name forage poisoning, which has been more or less in favor ever since. There are certain objections to this term, principally from the fact that it may suggest a form of poisoning produced by vegetation that is specifically poisonous, such as lupines, loco, larkspur, etc., or by ordinary forage that is poisonous of itself. This, however, was not the intention of Pearson, for by his analogy to meat poisoning it is evident that he did not wish to convey the impression that all forage was poisonous any more than all meat is poisonous. But when meat becomes contaminated with pathogenic bacteria, such as the *Bacillus enteritidis*, *B. botulinis*, etc., such meat is dangerous to man in the same manner that ordinary forage contaminated with certain unknown infective agents becomes dangerous to horses and produces forage poisoning. In other words, the forage is the carrier and not the primary factor in the disease. On the other hand, this term has a direct advantage in being readily understood in popular usage and in conveying to the layman's mind that an absolute change in feed is essential.

After years of study and experimentation it is the consensus of opinion of practically all investigators that the disease can be controlled effectively only by a total change of feed and forage; in other words, by preventive measures and not by medicinal treatment. That there is direct connection between ingestion of green forage, exposed pasturage, newly cut hay and fodder, and the development of the disease is quite obvious, and that the ingestion of such forage when contaminated is the most important factor is equally obvious, as almost 100 per cent of the cases in Kansas and over 95 per cent of the cases in Nebraska of which we have any record were maintained all or part of the time under such conditions. Even such negative history is not always dependable, as the owner on one farm informed the writer positively that the dead horses had eaten nothing except old hay and grain, but when notice was taken of the closely cropped grass in an adjacent pasture he innocently remarked that he always turned the work horses into the pasture over night. In fact in some sections "pasture disease" is the designation for this malady.

Other names which have been given to this affection are epizootic encephalo-myelitis, meningo-encephalitis and meningo-myelitis, enzootic cerebritis, leuco-encephalitis, etc., but the writer prefers the old-fashioned terms cerebrospinal meningitis for the scientific term and "blind staggers" for the lay term. That the symptom of staggering is one of the most common mainfestations of the disease is shown by the clinical observations of Schmidt, who has made a close study of 415 cases, 377 of which developed staggering symptoms while standing or walking. The only symptom which occurred more frequently was the loss of appetite appearing in 410 animals, while the symptoms next in prominence were grinding of the teeth, which was observed in 349 cases, and difficulty in swallowing, which occurred in 335 cases.

ETIOLOGY.

Unfortunately no specific bacteria, fungus, virus, or other toxic principle has yet been found which can be considered as the cause of cerebrospinal meningitis in the horse. It is quite true that bacteriological investigation has given us a number of different organisms by an equal number of different investigators, each of whom has thought his particular organism to be the causative agent of the disease; but the fact remains that the four rules laid down by Koch have not been met with sufficient regularity to make the results satisfactory to the disinterested worker. Further investigations are necessary to decide which, if any, of the reported organisms is the true cause of the disease. That the disease may not have an etiological entity has been suggested by Weichselbaum, Hutyra, and Marek. This would seem quite probable if all the claims for the following different etiological factors were to be accepted. For instance, Siedamgrotsky and Schlegel incriminated a micrococcus as the cause of the disease. On the other hand, Johne found diplococci in the cerebrospinal fluid which he termed *Diplococcus intracellularis equi*. Again, Ostertag recovered streptococci in short chains from the blood, liver, urine, and brain of affected horses. These organisms he termed Borna streptococci. Harrison of Canada isolated a streptococcus from the brains of horses affected with cerebrospinal meningitis which was quite similar to Ostertag's, although it differed in forming capsules, staining by Gram's method, refusing to grow well on gelatin, and in proving virulent for laboratory animals. In Minnesota, Wilson and Brimhall have also incriminated a diplococcus as the cause of cerebrospinal meningitis of horses, cattle, sheep, and pigs, and proved it to be the *Diplococcus pneumoniæ* of Frankel. They likewise claimed to have isolated the *Micrococcus intracellularis meningitidis* of Weichselbaum from the central nervous system of a cow showing symptoms of spinal meningitis. This latter organism

is also reported to have been found by Christiana in primary sporadic meningitis in the horse and in a goat.

The remarkable part of all the above investigations is that each author considers his particular organism as the etiological factor of the disease, and the majority of these writers believe they have succeeded in producing the disease in horses by the inoculation of these differing agents. Some of these positive results are readily explained by the large quantity of turbid fluid injected under the dura. The inoculation of 5 and 10 c. c. doses of a heavy emulsion of any organism is likely to produce an irritation, and the inflammation set up by such foreign material will necessarily produce exudation with accompanying mechanical pressure, so that it is not surprising to read in the post-mortem notes of some of these cases that the meninges bulged through the opening on cutting through the bones of the skull.

Schmidt, of Dresden, is of the opinion that the nature of the infectious principle is not settled, and believes that the cocci and diplococci which have been described as causative factors will in future be deprived of their pathogenic relationship.

In two outbreaks of forage poisoning investigated by Moore, of Cornell, one gave negative results from a bacteriological standpoint, while in the other pure cultures of the colon bacillus were obtained from the brain.

Grimm, working in Zwick's laboratory in Berlin, isolated streptococci from horses affected with head disease or staggers, which were not essentially different from the Borna streptococci of Ostertag. Owing to the regularity with which these cocci were taken from the brains of horses with "head disease"—cocci which Grimm stated possessed slight, if any, properties necessary to make them causal factors of disease—the question arose whether the same microorganisms are not also found in the brains of healthy horses. Grimm obtained the heads of 10 horses which were killed at the Zoological Garden for the animals, and which were by examination found to be free from any indication of cerebrospinal meningitis. In the brains of these healthy horses he found cocci (staphylococci and streptococci), although cultures were made within a few hours after death, and at least one strain has shown many similarities to the streptococcus found by Ostertag.

These results of Grimm's work are very similar to the results of the Bureau of Animal Industry. In horses which have died of forage poisoning it is not a difficult task to recover various forms of cocci; in fact, too many forms to make them all of etiological significance, while in those cases which have been killed in the late stages of the disease it is of common occurrence to have all the culture media inoculated with the various tissues remain sterile. On the other hand, we found micrococci, diplococci, streptococci, and staphylococci so frequently

in the brains of horses which have died of dourine, swamp fever, influenza, etc., that we have come to consider these organisms as representing an agonal invasion from the intestines without causal connection with any definite disease. Like Grimm, we have found some of these same cocci in the brains of horses that died of forage poisoning, and we have also recovered other species, all of which have been inoculated into experiment horses by various methods, including intravenous, subcutaneous, subdural, and intralumbar injection, as well as by spraying the nasal mucous membrane, with the result that two horses died following a nasal douche and a subdural injection, respectively, of a pure culture of two different cocci. The postmortem on the former showed death to have been due to a strangulated intestine, while the second animal died suddenly without evincing any characteristic symptoms, although extremely nervous. Postmortem examination showed an absence of any pathological lesions posterior to the brain. The dura mater was inflamed and distended with a yellowish exudate. The veins and capillaries of the cerebrum were dilated and engorged with blood, while the third ventricle contained a tumor the size of a walnut. Although the same organism which was injected was recovered from the brain tissue, other horses injected with the recovered culture have continued to remain in a healthy condition.

With the view of obtaining additional information regarding the significance of these various cocci to the disease in question, an antigen was prepared from a culture of each organism and tested against the blood serum obtained from affected horses in the field for complement fixation and agglutination as in glanders. In no case was a positive reaction to these tests obtained by the use of any of the antigens prepared from the different cocci isolated from diseased horses. In this connection it may be noted that from the number of affections of the horse produced by coccoid organisms, this animal appears to be particularly susceptible to their action.

Another cause has been suggested for this disease in the finding of nuclear inclusions by Joest and Degen in the nerve cells of the hippocampus. These inclusions are similar to the Negri bodies of rabies, and are rounded or oval in shape, staining intensely with eosin. A large number of brains from affected horses have been examined in our laboratory for these bodies, but thus far with negative results, although the same technique applied to the brains of rabid animals brings out the Negri bodies with great clearness.

There remains one widely accepted theory as to the causation of the disease which must be given consideration, namely, fungi on the feed. While most investigators have obtained negative results when feeding experiment animals upon moldy feed, some few have reproduced the disease by such feeding. Thus, Mayo reports that a

colt fed experimentally upon some of the moldy corn, which was held responsible for the serious outbreak in Kansas in 1890, developed the disease and died on the twenty-sixth day. Again, the Kansas outbreak of 1906 was said by Haslam to have been produced by immature ears of corn infected by molds, although the exact mold was not discovered. By feeding horses upon this immature corn badly infected with molds, typical fatal cases of staggers were produced in four out of seven horses. Haslam also records the fact that severe losses of horses have occurred in other States when the grasses in the pastures became moldy. Klimmer, commenting upon the negative results obtained in experiments with moldy feed, asserts that the numerous losses occurring from the feeding of such material indicates the probability that the experiments were not sufficiently extensive from which to draw conclusions, and believes that the use of such feed should be discouraged. Among other writers who have attributed the disease to toxic fungi are Michener, Trumbower, and Harbaugh. The latter investigated the serious outbreak of this disease which occurred in Virginia and North Carolina in 1886, and claimed that every case of the disease could be traced directly to moldy feed.

This theory of toxic fungi is not antagonistic to the facts in many of the best observed outbreaks, and knowing that fungi vary greatly in growth and in the elimination of various products under different climatic conditions, we may explain the irregularity of the symptoms as well as the occurrence of the disease under what may appear to be identical conditions. Thus Ceni of Italy states that molds are capable of producing poisons, but only at certain stages of their growth, and at other times they are entirely inactive. A case of this character was investigated by this bureau several years ago in an outbreak among the United States Army horses at an encampment in Pennsylvania. Many horses had died of cerebrospinal meningitis as a result of eating moldy baled hay, and as soon as the hay was eliminated the deaths ceased. Other horses in the vicinity not fed upon this hay failed to contract the disease. At the suggestion of State Veterinarian Marshall the bales were opened and exposed to the sun for three or four weeks, after which time this hay was fed sparingly at first and later in usual quantities without producing any ill effect. Forage poisoning therefore seems to be an auto-intoxication rather than an infection, and due to certain chemical poisons or toxins formed by organismal activity. These toxins may be present when the forage is taken into the body or formed in the gastro-intestinal canal, and, therefore, the disease is a specific form of auto-intoxication. The nature of the substance which causes these harmful changes or the poisonous bodies that are formed remain unknown.

On account of this very old and very plausible theory so often advanced, that the disease is due to toxic substances existing in damaged grain and fodder, a number of species of fungi were isolated during the past year from damaged corn and forage and grown on a sterilized corn medium or alfalfa infusion in an effort to produce some toxic substance that would create disease when fed to horses. The pure cultures were allowed to grow for periods of one month's duration, in flasks containing 250 cubic centimeters of the nutrient medium, and the contents of one flask were fed each day for periods of 30 days, along with a sufficient quantity of sound corn and hay to make a normal ration; but no symptoms have thus far developed in the experiment animals, although only about one-half of the number of pure cultures isolated have thus far been used in this experiment.

It is possible that laboratory conditions alone can not be made to parallel sufficiently close those which exist naturally in the growing plants, and that toxic substances which might be produced in a natural state would not be generated in a corn-meal medium in the laboratory. The by-products of the growth of both fungi and bacteria on corn and forage should certainly receive more consideration in future work. In view of the above information it must appear to the unbiased mind that the cause of forage poisoning remains an obscure and puzzling problem.

OCCURRENCE.

Like cerebrospinal meningitis of man, forage poisoning occurs in sporadic as well as enzootic and epizootic forms. The sporadic cases occur either in different localities from the epizootic outbreaks or in such sparse numbers as not to amount to an enzootic. Thus the outbreaks are quite variable in extent and severity. Sometimes they become very widespread, causing heavy losses, as in the recent outbreak in Kansas and Nebraska, while at other times there are only sporadic cases. Liebener believes that the development of the cause of the disease in Germany is favored by the rainfalls and warmth of the earth during summer and autumn. No conclusive evidence has ever been presented to indicate that the disease is ever transmitted directly from one horse to another. Sick animals have been placed alongside of susceptible horses in the same stable without conveying the disease to the latter, and healthy horses have been placed in stalls previously occupied by animals which died of the disease, and have eaten from the same mangers without previous disinfection, but in no case has the disease been transmitted in this manner. In the recent outbreak in Kansas it was quite noticeable that livery and other work horses were not affected so long as they were fed on clean, dry forage, although they were constantly exposed

to the disease by coming in contact with diseased horses. For instance, Dr. Herman Busman, who was in charge of the Kansas field force of veterinarians of the Bureau of Animal Industry, reports a case where horses were kept in adjoining corrals separated only by a wire fence. Those on one side were fed on green forage and recently cut cane, and died from the disease, while those on the other side were fed dry feed and not one became sick. He also reports a similar occurrence in a livery barn where the horses had been fed on clean, dry feed without sickness, but when fresh cut bottom-land hay was substituted for the former feed the horses became sick within a few days. Another similar instance was reported by Dr. E. T. Davison, in charge of the bureau's field force in Nebraska, in the case of a farmer who owned a work team that was strictly barn fed. While attending the State Fair at Lincoln these horses were turned out on pasture for two days and both horses came down with the disease on the fourth and fifth days, respectively, after being taken off the pasture.

It is such cases as these that have incriminated the forage and caused the disease to be known as "pasture disease" in some localities. Indeed some veterinarians report that all the animals affected had been on pasture, or, having been removed from pasture, had been fed on recent cuttings of alfalfa, prairie hay, cane, or kafir corn, while no cases came under observation where the animals had been on dry feed all summer.

A long period of dry weather followed by rainfall with considerable humidity and heat seems to favor the development and dissemination of the disease. The period from August 1 to October 1, 1912, presented exceptional climatic conditions in western Kansas and southern Nebraska, and it was observed that crops cut and cured before this date could be fed with impunity. During the first week in August a heavy rainfall started in Kansas and nearly twice the usual amount was recorded, falling mostly during the night and soaking in. This was followed by very high temperatures, the 17 days from August 23 to September 9 being the hottest series of days on record in Dodge City. There were also more than the usual number of cloudy or partly cloudy days with high relative humidities. The dew point was reached early at night and the deposit of dew was abundant, which is uncommon in that section. High humidities certainly continued throughout the day among the grasses near the soil. These grasses, which usually cure into hay on the root, became dotted with both parasitic and saprophytic fungi. Water holes, draws, and buffalo wallows remained filled with water throughout most of the period. During the latter part of September frosts occurred, accompanied not only by cooler weather but with lower

humidity, which are the significant factors in the subsidence of the disease, and after the first week in October the disease practically disappeared. Since then many owners have placed their horses back on the same pastures used during the serious stages of the disease and there has been no ill effect noted. This would indicate that there are good reasons to believe the forage is no longer in condition to produce the disease and hence its use is safe, as in the case of the Pennsylvania baled hay previously mentioned.

Somewhat similar conditions of climate obtained in Nebraska during the prevalence of the disease, but on September 25 a killing frost was recorded, followed by several light frosts and a reduction in the relative humidity. After this time the disease rapidly subsided and finally disappeared. There is not much question that some of this infected forage has been baled and shipped to various points, and it is therefore not unlikely that sporadic cases of the disease will appear in these sections under favorable climatic conditions.

In this connection, attention should be called to the marked prevalence last summer and fall of the disease of cattle known as mycotic stomatitis, which simulates the foot-and-mouth disease of Europe and is caused also by contaminated forage. This disease first appeared in Florida and spread over Georgia, North and South Carolina, Tennessee, Kentucky, Virginia, Maryland, and into Pennsylvania. The climatic conditions were evidently appropriate for the development of the causative agent on forage, and as soon as the animals were brought out of the pastures and stall fed, the disease immediately subsided.

SYMPTOMS AND LESIONS.

In most of the cases disturbance of the appetite, depression, and weakness are the first manifestations observed, although all the symptoms vary within wide limits.

Very soon the characteristic symptoms of the disease appear. There is trouble in swallowing, drooping of the head and sleepiness, which may give way to excitement and attacks of vertigo. An impairment of vision is noted, with loss of coordination, resulting in a staggering gait or reeling while standing. There is muscular twitching, cramp of certain muscles, chiefly of the neck and flanks, and grinding of the teeth. Sometimes colicky pains are noted. If in an open space, the animal will walk in a circle, sometimes to the right, at other times to the left, and will try to push through any obstacle with which he comes in contact. In the stable he will press his head against the stall or rest it on the manger. Sometimes he will crowd backward or sidewise until he gets in a corner and remains there. If the temperature is taken at the beginning of the disease it will be

found to be from 103° to 107° F., but within 24 hours the temperature gradually falls until it reaches normal and then becomes subnormal. The pulse is from 40 to 90 and weak, while the respirations are fluctuating from normal to as high as 48 per minute. There may or may not be drooling of saliva, depending on the extent of the paralysis of the pharynx. The animal is often down on the second or third day and may or may not get up when urged to do so. While down he will go through automatic-like movements of pacing or walking, resulting in acceleration of the pulse and respiration. At this time the legs are held out stiffly and parallel to the ground. The hind legs of many of these animals that have gone down are paralyzed and there is loss of sensation of the skin of these parts. The expired air is extremely fetid, and there may be a croupous-like deposit of the throat, which has caused the name "putrid sore throat." The conjunctiva may show injected blood vessels or petechiæ on a yellowish-tinted background. Coma or somnolence may be marked in animals going down within the first few days. Those which remain standing may become violent or delirious, but ordinarily the horse is tractable and easily managed. Death usually occurs in from 4 to 8 days, although in the acute form death may follow within 10 or 12 hours after the first symptoms are observed, while in chronic cases the disease may last 2 or 3 weeks. The prognosis is very unfavorable, as 85 to 90 per cent of the affected animals die, in the beginning of the outbreak, but later the cases become milder with a consequent drop in the mortality.

On post-mortem the amount of lesions observable to the naked eye is in marked contrast to the severity of the symptoms noted. The pharynx and larynx are inflamed in many cases, and sometimes coated with a yellowish white glutinous deposit, extending at times over the tongue and occasionally a little way down the trachea. The lungs are normal, except from complications following drenching or recumbence for a long period. The heart is usually normal in appearance, except an occasional cluster of petechiæ on the epicardium, while the blood is dark and firmly coagulated. The mucosa of the stomach indicates a subacute gastritis, while occasionally an erosion is noted. An edematous, gelatinous infiltration is observed in the submucosa of such cases. The first few inches of the small intestines likewise may show slight inflammation in certain cases, while in others it is quite severe; otherwise the digestive tract appears normal, excluding the presence of varying numbers of bots, *Strongylus vulgatus*, and a few other nematodes. The liver is congested and swollen in some cases, while it appears normal in others. The spleen is, as a rule, normal, and at times the kidneys are slightly congested. The bladder is often distended with dark-colored urine, and occasionally a marked cystitis has been observed. The adipose tissue

throughout the carcass may show a pronounced icteric appearance in certain cases. On removing the bones of the skull the brain appears to be normal macroscopically in a few instances, but in most cases the veins and capillaries of the meninges of the cerebrum, cerebellum, and occasionally the medulla are distinctly dilated and engorged, and in a few cases there are pronounced lesions of a leptomeningitis. An excessive amount of cerebrospinal fluid is present in most of the cases. On the floor of the lateral ventricles of several brains there was noted a slight softening due to hemorrhages into the brain substance. There is always an abundance of fluid in the subarachnoid spaces, ventricles, and at the base of the brain, usually of the color of diabetic urine, and containing a limited amount of flocculi, but in a few cases it was slightly blood tinged. The spinal cord was not found involved in the few cases examined.

A comparative microscopic examination of the brains of horses which died in Kansas, New Jersey, Maryland, and Virginia this year with those of horses from previous outbreaks showed the same characteristic perivascular round-cell infiltration, especially in the olfactory lobe and the hippocampus. The pia mater showed an increased amount of connective tissue with dense round-cell infiltration which extended into the adjacent cortical portion of the cerebrum. The capillary blood vessels were engorged with cells and their walls were greatly infiltrated. Limited areas of leucocytic infiltration and small hemorrhages in the brain tissue were not infrequently observed. No cellular inclusions in the ganglionic cells were detected after prolonged examination.

TREATMENT.

One attack of the disease does not confer immunity. Horses have been observed which have recovered from two attacks, and still others that recovered from the first but died as a result of the second attack.

Inasmuch as a natural immunity does not appear after an attack of cerebrospinal meningitis, it might be anticipated that serum of recovered cases would possess neither curative nor prophylactic qualities. Nevertheless, experiments were made along these lines with serum from recovered cases, but without any positive results. Similar investigations have been conducted by others in Europe with precisely the same results. With the tendency of the disease to produce pathological lesions in the central nervous system, it seems scarcely imaginable that a medicinal remedy will be found to heal these foci, and even where recovery takes place there is likely to remain some considerable disturbance in the functions, as blindness, partial paralysis, dumbness, etc. Indeed, when the disease once becomes established in an animal, drugs seem to lose their physio-

logical action. Therefore, with all the previously mentioned facts before us, it is evident that the first principle in the treatment of this disease is prevention, which consists in the exercise of proper care in feeding only clean, well-cured forage and grain and pure water from an uncontaminated source. These measures when faithfully carried out check the development of additional cases of the disease upon the affected premises.

While medicinal treatment has proved unsatisfactory in the vast majority of cases, nevertheless the first indication is to clean out the digestive tract thoroughly, and to accomplish this prompt measures must be used early in the disease. Active and concentrated remedies should be given, preferably subcutaneously or intravenously, owing to the great difficulty in swallowing, even in the early stage. Arecolin in one-half grain doses, subcutaneously, has given as much satisfaction as any other drug. After purging the animal the treatment is mostly symptomatic. Intestinal disinfectants, particularly calomel, salol, and salicylic acid, have been recommended, and mild antiseptic mouth washes are advisable. Antipyretics are of doubtful value, as better results are obtained, if the temperature is high, by copious cold-water injections. An ice pack applied to the head is beneficial in case of marked psychic disturbance. One-ounce doses of chloral hydrate per rectum should be given if the patient is violent or muscular spasms are severe. If the temperature becomes subnormal, the animal should be warmly blanketed, and if much weakness is shown this should be combated with stimulants, such as strychnin, camphor, alcohol, atropin, or aromatic spirits of ammonia. Early in the disease urotropin (hexamethylenamin) in doses of 25 grains, dissolved in water and given by the mouth every two hours, appeared to have been responsible for the recovery of some cases of the malady. During convalescence the usual tonic treatment is indicated.

Many of the so-called "cures" made their reputation at the time the outbreak was abating and when noninterference was proved to be equally effective. One of the most unpleasant developments of the outbreak in 1912 was the great amount of "faking," which seemed to be the only contagious feature connected with the disease. All kinds of drug specifics, serums, and vaccines developed like mushrooms and were exploited in almost every community devastated by the disease. Many tainted dollars were obtained from the suffering horse owners, who grasped at every newly advanced treatment like drowning men clutching at straws. In Nebraska, blackleg vaccine was reported to have been used as a preventive on at least 1,600 horses, and nearly 1,500 of them are said to have died as a direct result of the vaccine. This feature is now being investigated by the Government.

Dr. Munn, of Kearney, Nebr., had apparently good success from the use of diphtheria antitoxin as a prophylactic agent, since not a single animal developed the disease out of over 500 injected. It may be with this treatment, as with others, that good results were due to the fact that the disease was on the wane before treatment was commenced, but no other line of treatment gave as good apparent results. Dr. Kaupp also reports in the Breeders' Gazette that only 1 horse died out of 900 inoculated with a diplo-streptococcic bacterin he prepared, but the injections were made so late in the outbreak that its value is still problematical, since thousands of horses in the affected area at this period failed to develop the disease, although they had received no preventive treatment whatsoever.

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